DASA-1483

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THE EFFECTS OF AMBIENT PRESSURE ON THE TOLERANCE OF MICE TO AIR BLAST

Edward G. Damon, Donald R. Richmond
and Clayton S. White

Presented before the
Symposium on the Biological Effects
of Blast and Shock
Headquarters Field Command
Defense Atomic Support Agency
Sandia Base, Albuquerque, New Mexico
April 20, 1963

Technical Progress Report
on
Contract No. DA-49-146-XZ-055

This work, an aspect of investigations dealing with the Biological Effects of Blast from Bombs, was supported by the Defense Atomic Support Agency of the Department of Defense.

Lovelace Foundation for Medical Education and Research
Albuquerque, New Mexico
March 1964

This report has been approved for open publication by The Office of the Assistant Secretary of Defense for Public Affairs.

FOREWORD

This report describes the development of appropriate apparatus and experiments to explore the relationship between the response of animals to air blast and the ambient pressure existing at the time of exposure. Specifically, the tolerance of mice to overpressure was determined using the expansion chamber of a specially modified shock tube in which the local pre-shot pressures were varied from 7 to 42 psi absolute.

The results and experience from this study will guide the design of appropriate hardware and the initiation of similar investigations, wherein larger animal species may be employed in the most economical manner. The ultimate aim of the work is the accurate prediction of human tolerance to air blast as a function of reduced pressure associated with altitude and increased pressure corresponding to various locations below sea level. Thus the findings have applicability in Aviation, Submarine and Environmental Medicine and are significant in air evacuation of blast-produced casualties, the care and therapy of blast injuries occurring underwater, inside submarines, aloft in aircraft and in other pressurized locations.

ABSTRACT

Mice were exposed to overpressures of "long" duration in the expansion chamber of an air-driven shock tube inside which the initial, preblast pressures were varied over sixfold. When the animals were held at the initial pressure for one hour following the blast before being returned to the ambient pressure of the laboratory, tolerance values, expressed as LD_{50}-1-hour gauge pressures, increased fourfold; they were 20.3, 31.0, 44.5, 55.4, and 91.8 psi for initial pressures of 7, 12, 18, 24, and 42 psia, respectively. When animals were returned to ambient level soon after blast exposure, the LD_{50} pressures were lower than the above values for initial pressures greater than ambient and higher for initial pressures lower than ambient. The feasibility of scaling biological blast effects as a function of altitude was discussed and one approach suggested by available empirical data was regarded as a promising, but tentative procedure.

ACKNOWLEDGMENTS

The able technical assistance of Mr. Charles S. Gaylord, Mr. Peter A. Betz, Mr. William Hicks, Mr. Donald E. Pratt, Mr. Dennis D. Branch, Mr. Kabby Mitchell, Jr., Mr. Keith G. Saunders, and Mr. Raymond T. Sanchez is greatly appreciated. Also, the aid of Mr. Robert A. Smith, who prepared illustrative material, Mr. Ray W. Albright, who carried out the probit analysis on a Bendix G-15 electronic computer, and Mrs. Maxine U. Thibert, who assisted with the illustrations, typed and edited this report, is acknowledged.

This report covers a portion of the work being presented by Mr. Edward G. Damon in a dissertation to be submitted to the University of New Mexico in partial fulfillment of the requirements for the Ph. D. degree.

Experiments described herein were conducted according to the Principles of Laboratory Animal Care as promulgated by the National Society for Medical Research.

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THE EFFECTS OF AMBIENT PRESSURE ON THE TOLERANCE OF MICE TO AIR BLAST

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INTRODUCTION

Although the relationships between biological response to air blast and various parameters of the pressure wave have been investigated in recent years,[1-4] very little is known about the effects of ambient pressure on mammalian tolerance to overpressure. Not only was attention called to this fact several years ago,[5] but for theoretical and commonsense reasons, it was predicted that the ambient pressure existing at the time an animal was loaded with a pressure pulse would be a significant parameter influencing biological response. Without question, it is important to know whether or not this speculation has validity, and if so, the magnitude of the effect, because human exposures to detonation-produced variations in the environment can and do occur at a variety of ambient pressures such as those existing at different elevations of terrain, at whatever levels above and below the earth's surface are available to man and at duty stations inside different manned vehicles and pressurized spaces wherever they may be.

The present investigation was undertaken to develop shock-tube and related techniques for exposing animals to air blast at different ambient pressures and to explore the tolerance of mice to "sharp"-rising overpressures of "long" duration as related to pre-shot ambient pressures ranging from a fraction of an atmosphere to several atmospheres.

METHODS

Shock Tube

A conventional, circular shock tube, 19 ft 6 in. long and 12 in. in diameter, was modified and used to expose mice to air blast at different ambient pressures. The tube had a wall thickness of 3/8 in., and as shown diagrammatically in Figure 1, was divided by a frangible diaphragm into a compression chamber 2 ft 6 in. long and a 17-ft expansion chamber. The latter was closed with an end-plate on which animal cages were mounted.

Appropriate pipes and valves, to allow pre- and post-shot control of the

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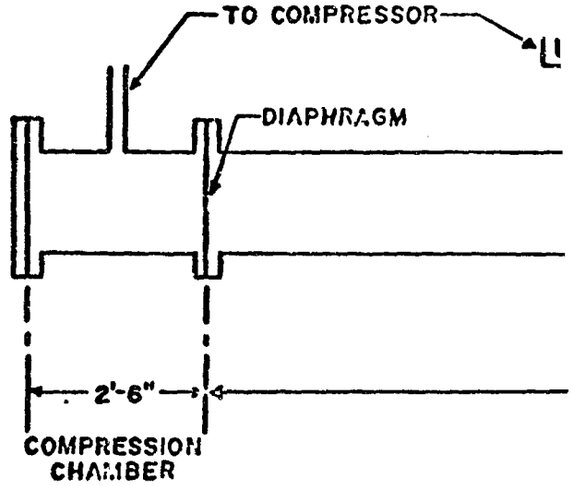
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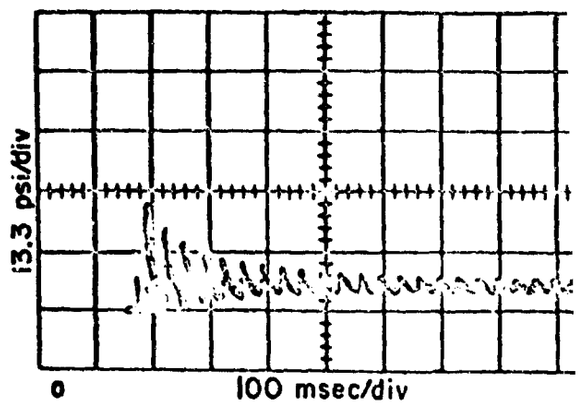
COMPRESSION CHAMBER


13.3 psi/div
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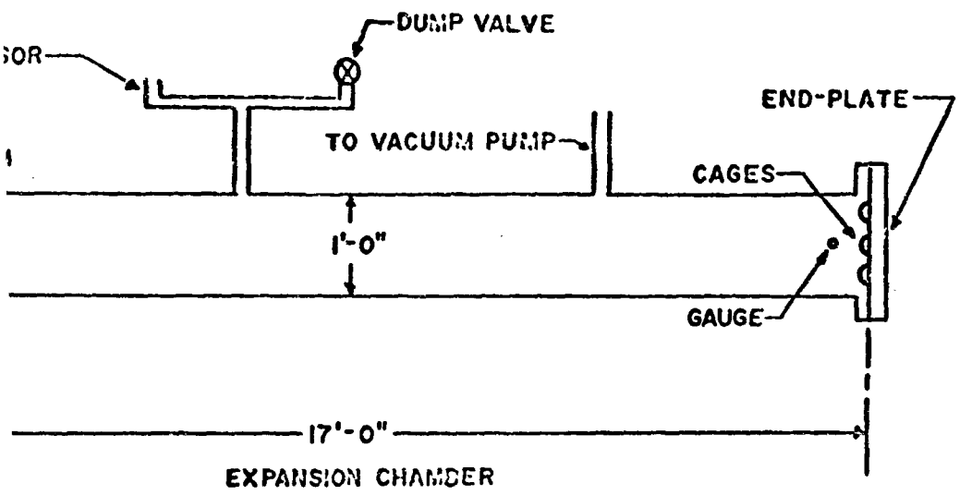
SHOCK TUBE LAYOUT



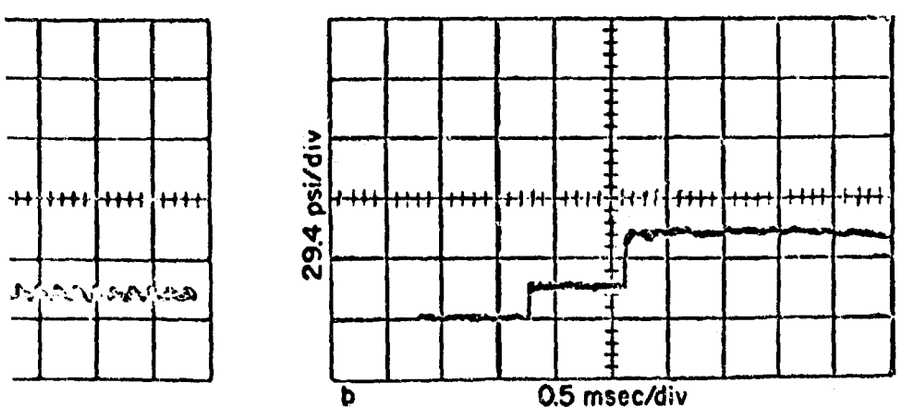


Figure 1

pressure inside the shock tube, were fitted to the expansion and compression chambers and multiple layers of Dupont Mylar plastic were employed as a diaphragm. Since experience proved that the Mylar sheets exhibited a consistent bursting pressure when tested on 12-in. tubes, different exposure pressures were achieved by varying the total thickness of the plastic and allowing each diaphragm to rupture spontaneously as the compression chamber was progressively pressurized.

Pressure-Time Measurements

On every test, the shock pressures were measured with piezoelectric gauges mounted side-on in the wall of the tube 6 in. upstream from the end-plate (Figure 1). Occasionally, gauges were also located on the end-plate to record the pressure-time wave form at the position of the animals. The piezoelectric transducers contained sensors of Lead Metaniobate (Model ST-2, Susquehanna Instruments, Bel Air, Maryland). Each signal from a pressure transducer was passed through a cathode follower and was displayed and photographically recorded on a cathode-ray oscilloscope. Details of the system and its calibration already have been reported.[4,6] Typical pressure-time oscillograms obtained with the gauge mounted side-on in the wall of the tube are presented in Figure 1.

The overpressure in the expansion chamber before and after each blast was measured by a Bourdon-type dial pressure gauge (Heise Bourdon Tube Co., Newton, Connecticut). A mercury manometer gave the pressure levels when the expansion chamber was partly evacuated. The time required to increase or decrease the pressure in the expansion chamber was carefully measured with a stopwatch and also checked on oscillograms obtained with Quartz piezoelectric transducers (Model PZ-4, Kistler Instrument Corporation, North Tonawanda, New York). The oscilloscopes were triggered so that the time to increase or release the pressure was recorded.

Figure 2 presents a comparison of the empirical, shock-tube calibration curve with theoretical data. The results indicate that the measured performance of the current hardware was within 10 per cent of that predicted by the theoretical relationships. Since this result is consistent with experience reported elsewhere[7,8] as characteristic of air-driven, conventional shock tubes, it indicates that the methods used to measure shock pressures were reliable at either reduced or elevated initial ambient pressures.

Animal Exposure

In all, 672 female mice of the Webster strain were employed. Their mean body weight was 19.7 g (standard error of the mean and range were ±0.84 and 16 - 24 g, respectively). Except where otherwise mentioned, three animals were exposed per shot. Each animal was oriented right-side-on to the incident shock in an individual, cylindrical, wire-mesh cage mounted against the end-plate. The diameter of the wire from which the cages were constructed was 1/16 in. and the inside diameter of the squares of the mesh was 1/4 inch. The cages were arranged 2 in. apart, one above the other. Since the end-plate of the tube was oriented normal to the incident shock wave, the

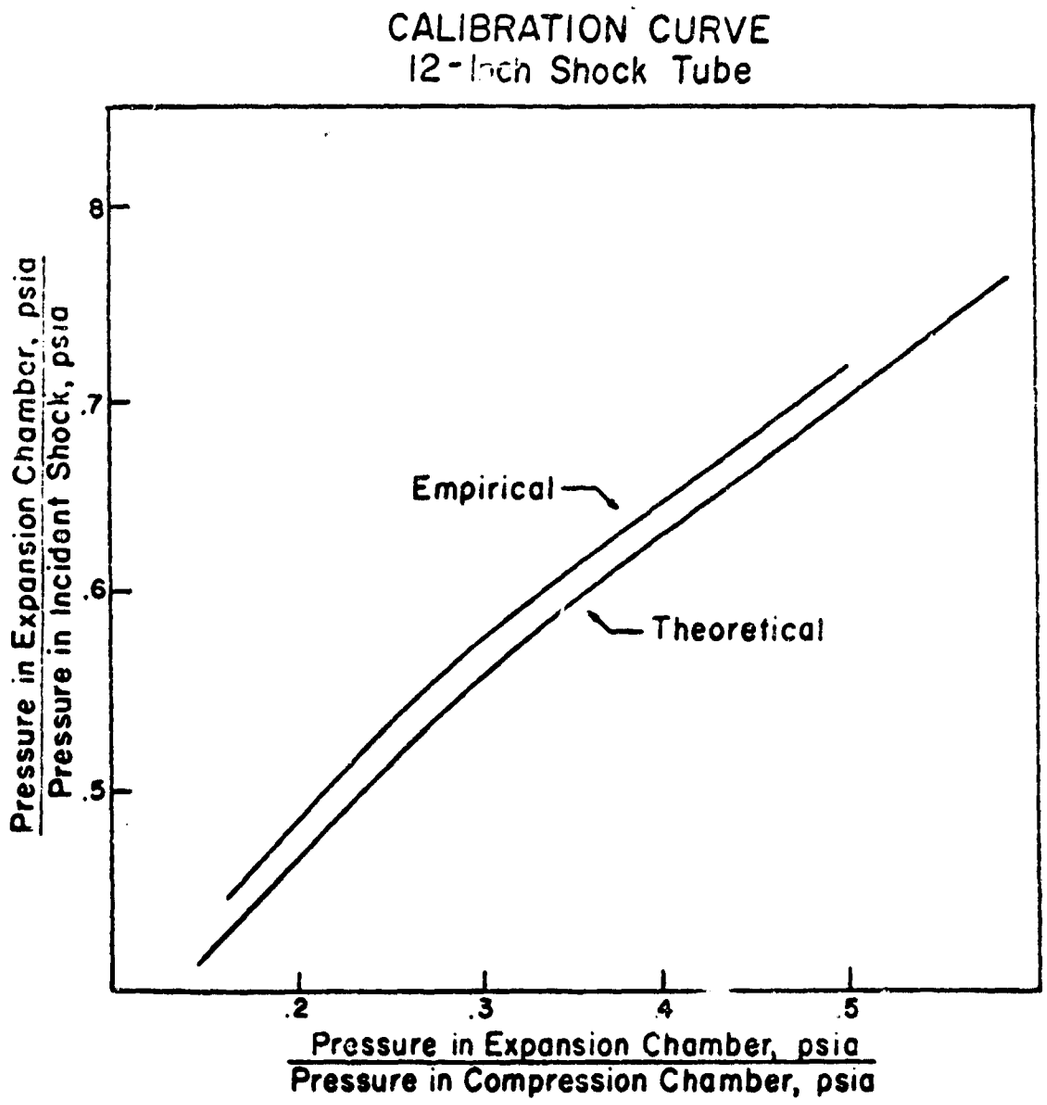


Fig. 2. Comparison of the calibration curve for the 12-in. shock tube with the theoretical curve for shock strength as a function of the starting pressure ratio (Bleakney, 1949).[7]

animals were subjected to the incident and the reflected shock almost simultaneously. Consequently, the air-blast dose was taken to be the maximal overpressure in the reflected shock. The duration of the positive phase of the primary blast wave was 16 - 20 msec, which is much longer than the "critical duration" for mice.[4] Following the first positive wave, the animals were subjected to a series of decreasing secondary pressure pulses resulting from the reflection of the shock wave from one end of the tube to the other. Pressure-time record "a" in Figure 1 is a typical oscillogram showing these multiple reflections.

Series I

Two hundred and seventy mice were exposed in groups to three levels of reflected shock pressures while at initial pressures of 7, 12, 18, 24, and 42 psia. Immediately after the blast, the pressure in the expansion chamber was quickly adjusted to the respective pre-shot level and then held for one hour before it was returned to ambient level.

The five overall pressure-time profiles for Series I experiments are illustrated in Figures 3a - 3e. Indicated are the times required to increase or decrease the pressure on the animals before and after the blast. For instance, Figure 3c shows that 25 seconds (t_1) were required to increase the pressure from the atmospheric ambient (P_o) of 12 psia to the initial pressure (P_i) of 18 psia in the expansion section. It was held for 78 seconds (t_2) before the blast. The duration of the blast wave itself was 0.016 seconds (t_3). After the blast, the pressure stayed at P_b (27 psia) for 2 seconds (t_4) before it could be reduced to the pre-shot level in 18 seconds (t_5). At the end of the 1-hour hold (t_6), the pressure was returned to ambient in 15 seconds (t_7).

Controls

Except for exposure to blast overpressures, 16 control animals were subjected to the pressure-time sequence illustrated in Figure 3e.

Series II

Two hundred and eighty-five mice were exposed to air blast at initial pressures of 7, 18, 30, 36, and 42 psia following the general procedures used in Series I animals, except they were returned to ambient immediately after blast exposure. The rates of pressure changes previous to and following the blast were kept similar to those in the Series I studies, except for the absence of the 1-hour hold period (t_6).

Controls

Fifteen Series II control animals were handled as Series I controls except they were not held for an hour at the pre-shot pressure (P_1) of 42 psia.

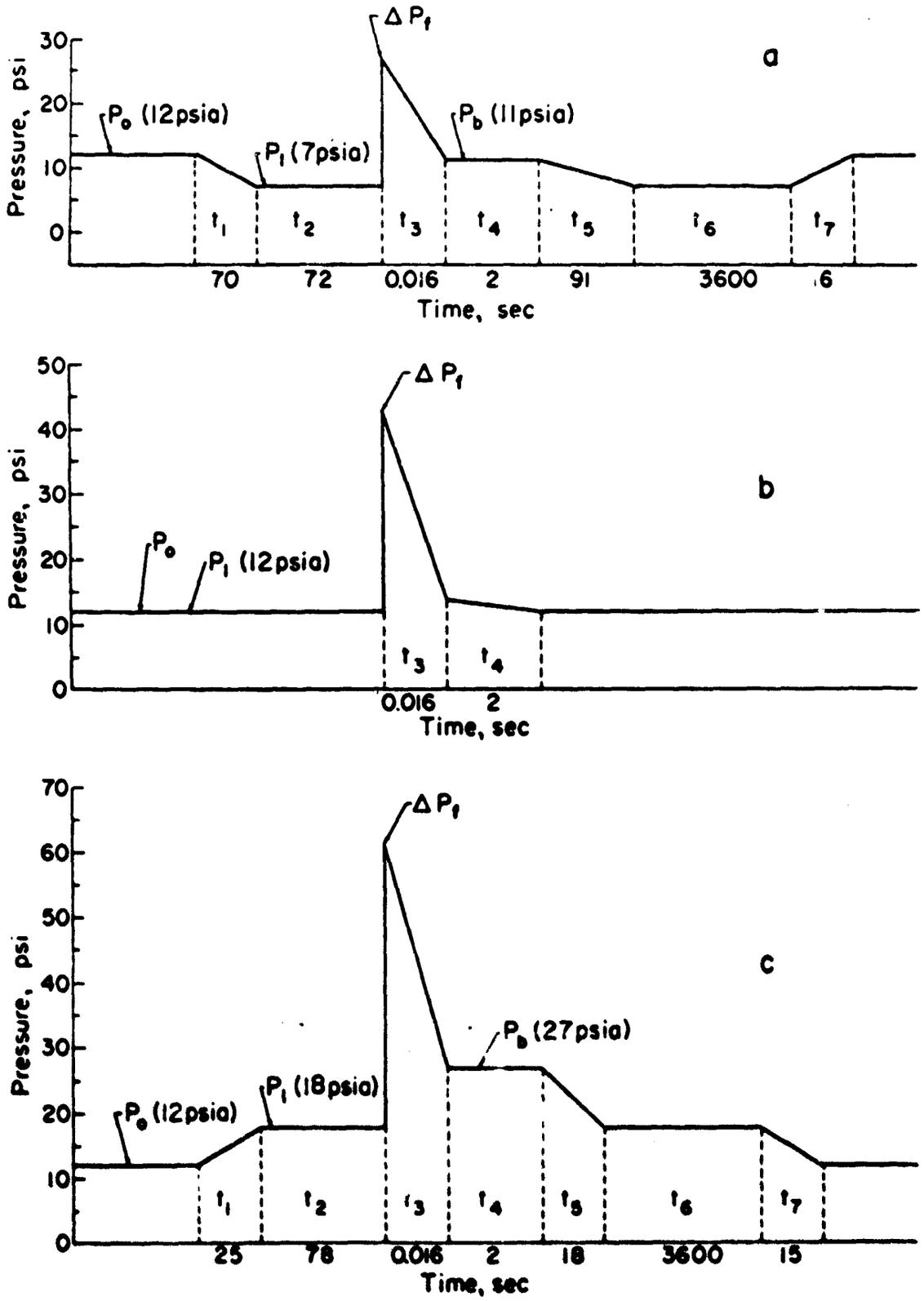
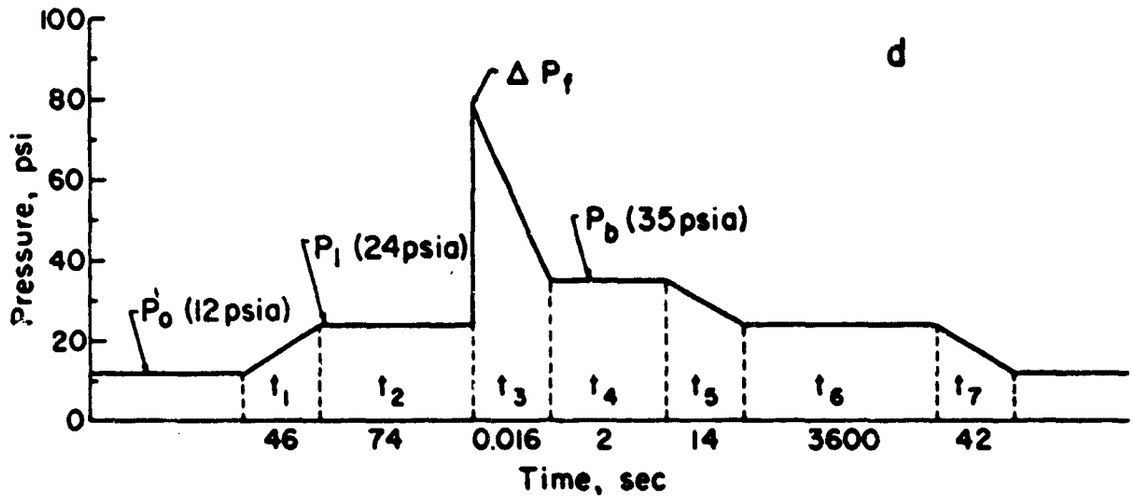


Fig. 3 (a-c). Overall pressure-time profiles for Series I.



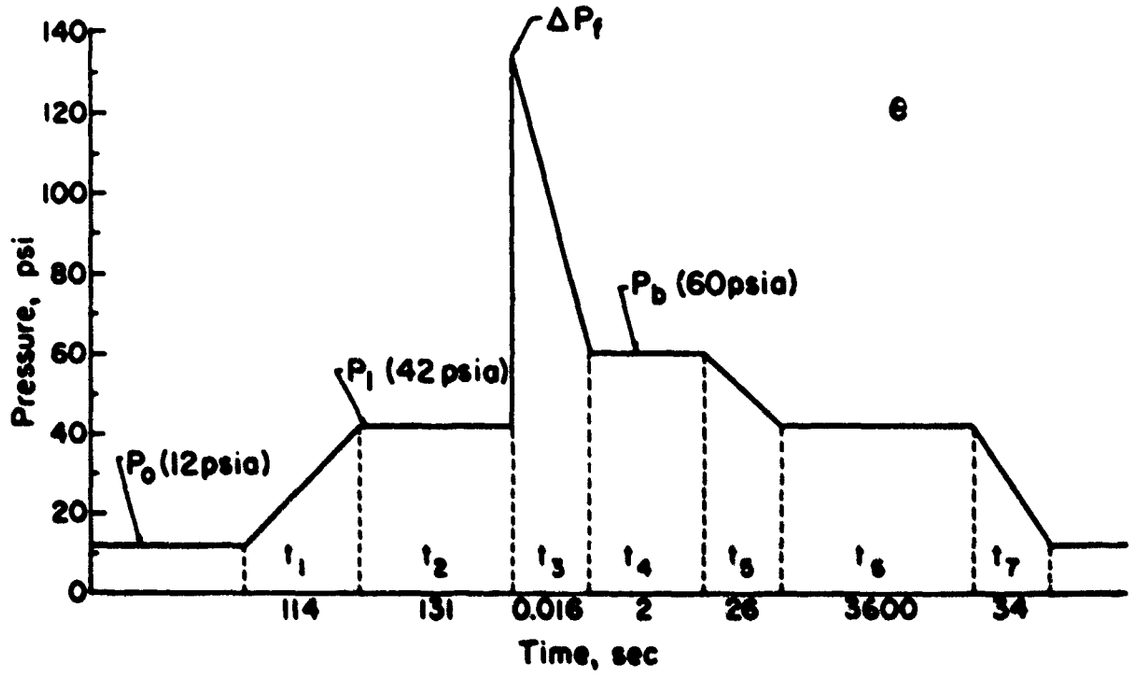


Fig. 3 (d-e). Overall pressure-time profiles for Series I.

Probit Analysis

Probit analysis was applied to the one-hour mortality data obtained from both experimental series.[9] Thus, the results presented refer to lethality within one hour following the blast.

In Series I, the total number of animals which were dead when first observed at the end of the 1-hour hold were recorded for the one-hour mortality. Of these, the number which exhibited signs of rigor mortis were also recorded. Since some of the animals could have died during the two minutes required for removing them from the tube following decompression, probit analysis was applied to both the total one-hour mortality data and the mortality data based on only those which exhibited signs of rigor mortis. Since there was no significant difference in the LD_{50} values computed from the two sets of data, only the results of the analysis of the total one-hour mortality data for Series I are presented.

RESULTS

Series I

Probit mortality curves relating the percentage dead in probit units to the log reflected pressure are presented in Figure 4 for the mice exposed at the five initial pressure levels in Series I. The probit regression lines were adjusted to an average slope since statistical tests revealed them to be essentially parallel at the 95-per cent fiducial limits.[9] The LD_{50} reflected shock pressures with their 95-per cent confidence limits and the probit regression equations' constants are listed in Table 1 along with the associated number of animals. As indicated in Table 1, the reflected pressure required for 50-per cent lethality rose as the initial pressures were increased. The LD_{50} pressures were 20.3, 31.0, 44.5, 55.3, and 91.8 psig when mice were exposed at initial pressures of 7, 12, 18, 24, and 42 psia, respectively. Each LD_{50} value differed significantly from the others at the 95-per cent confidence level. Actually, the LD_{50} values increased linearly with increasing initial pressures. A Bendix G-15 computer was programmed to fit a regression of the form, $\log y = a + b \log x$, to the data. Figure 5 presents the regression and a log-log plot of the data.

Table 2 compares the LD_{50} values in terms of reflected overpressure (psig) and atmospheres (atm) of the initial pressure ($\Delta P/P_i$). As noted, the LD_{50} pressure ratio ranged from 2.90 to 2.19 for initial pressures of 7 to 42 psia, respectively. Thus, in terms of atm of the initial pressure, biological tolerance decreased somewhat with increases in initial pressure.

Series II

The results of the probit analysis of the one-hour mortality data from the series in which the animals were returned to ambient immediately after

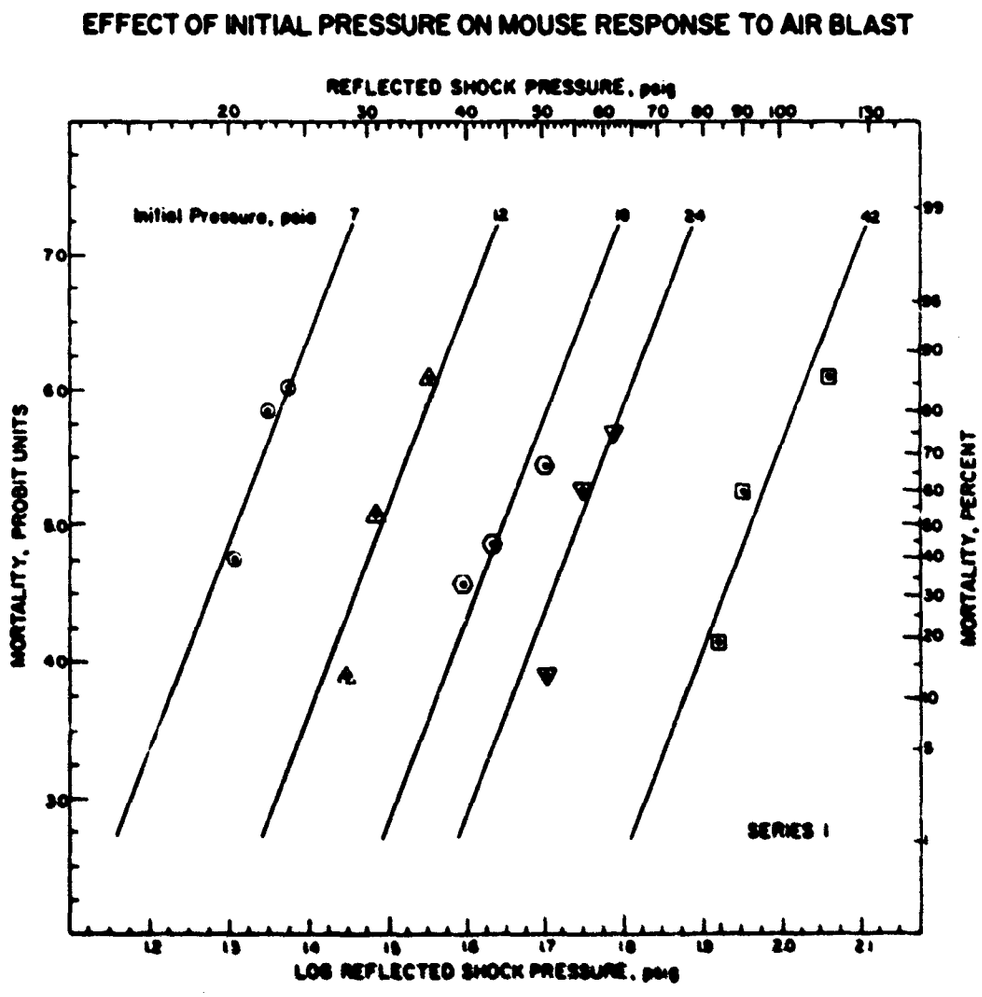


Fig. 4. Probit regression lines relating the percent mortality in probit units to the log of the reflected shock pressures for mice subjected to air blast at different initial air pressures.

TABLE 1

RESULTS OF PROBIT ANALYSIS OF THE SERIES I DATA

Initial Pressure, psia	Number of Animals	LD_{50}-1-hour Reflected Pressure (ΔP), psig	Probit Equation Constants	
			intercept, a	slope, b
7	60	20.3 (19.0-21.5)*	-14.481	14.889**
12	45	31.0 (29.3-33.3)	-17.254	14.889
18	48	44.5 (41.9-47.4)	-19.543	14.889
24	60	55.3 (52.4-58.3)	-20.948	14.889
42	57	91.8 (86.1-98.3)	-24.225	14.889
Total	270			

*Numbers in parentheses are the 95-percent confidence limits.
**Standard deviation of the slope constant, b - ±2.154.

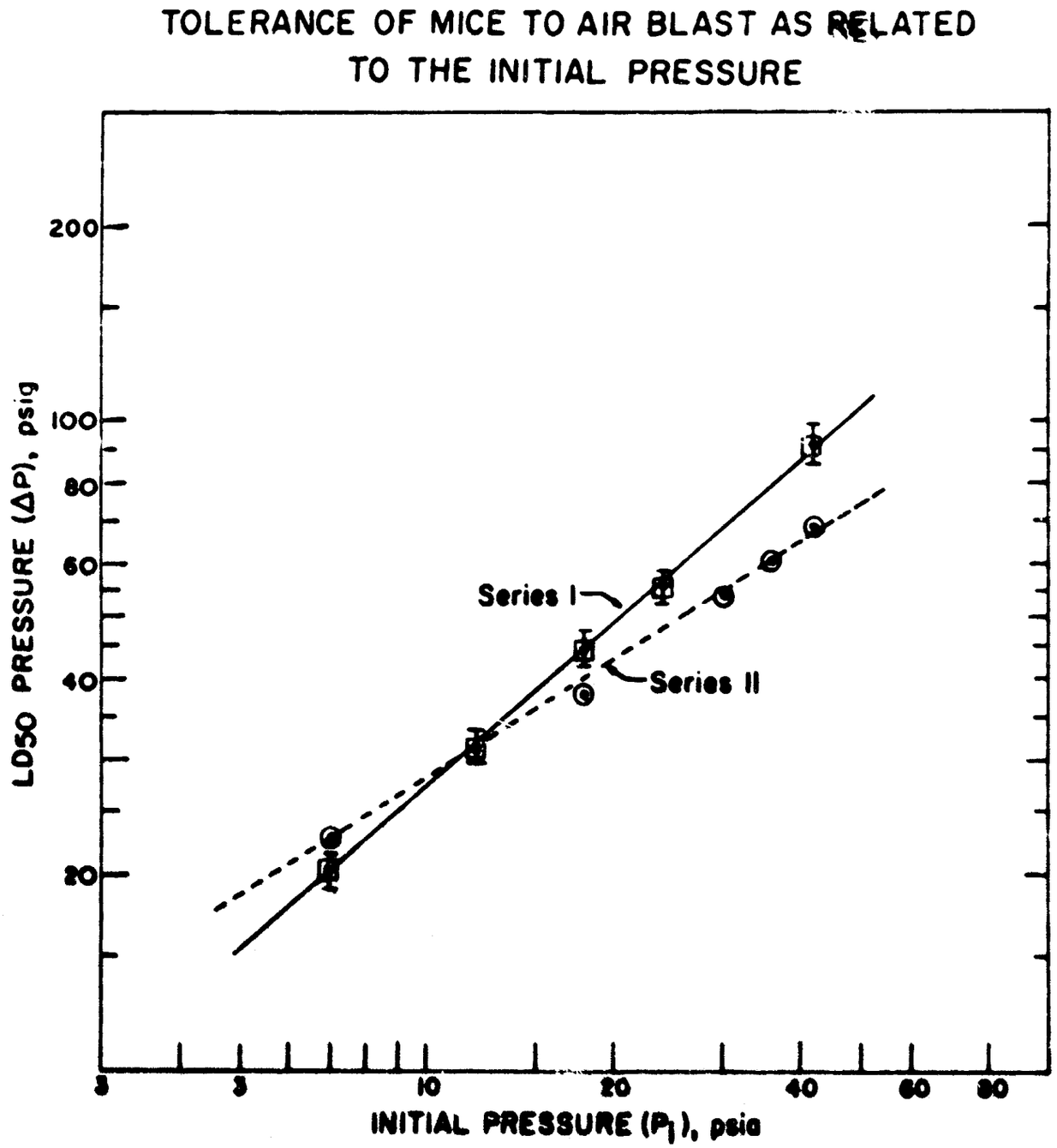


Fig. 5. LD_{50}-1-hour overpressure as a function of the initial pressure at exposure. Regression equations:
Series I, $\log (LD_{50}) = 0.590 + 0.842 \log (P_i)$;
Series II, $\log (LD_{50}) = 0.832 + 0.611 \log (P_i)$.

TABLE 2

COMPARISON OF LD_{50} VALUES

Initial Pressure, P_i, psia	LD_{50}-1-Hour Overpressure	
	ΔP, psig	atm*($\Delta P/P_i$)
7	20.3	2.90
12	31.2	2.60
18	44.5	2.47
24	55.3	2.30
42	91.8	2.19
	Average	2.49

*Atmospheres of the initial pressure.

blast exposure are presented in Table 3. The LD_{50} reflected shock pressures were 22.7, 37.9, 53.6, 61.3, and 68.4 psig for initial pressures of 7, 18, 30, 36, and 42 psia, respectively. As illustrated in Figure 5, the LD_{50} values were below those of Series I at initial pressures greater than ambient and above them for initial pressures less than ambient.

Controls

Results of control experiments revealed that the most rigorous combinations of decompression or compression, hold, and release of pressure (without the blast) encountered in this study, by themselves, produced neither deaths nor noticeable injury in mice. For instance, groups of animals were compressed to 67 psia in 225 seconds, held at that level for 2 minutes, and then returned to 42 psia and held for one hour, after which the pressure was reduced to 12 psia in 34 seconds. In addition, mice were compressed to 67 psia in 225 seconds, held there for 2 minutes, and then returned to 12 psia in 56 seconds.

DISCUSSION

This study, designed to explore the significance of ambient pressure on blast tolerance, shows an unequivocal increase in resistance to over-

TABLE 3

RESULTS OF PROBIT ANALYSIS OF THE SERIES II DATA

Initial Pressure, psia	Number of Animals	LD_{50}-1-Hour Reflected Pressure (ΔP), psig	Probit Equation Constants	
			intercept, a	slope, b
7	45	22.7 (21.0-24.6)*	-18.805	17.554**
18	69	37.9 (35.2-41.2)	-22.717	17.554
30	45	53.6 (49.4-58.7)	-25.359	17.554
36	57	61.3 (55.7-67.2)	-26.379	17.554
42	69	68.4 (64.2-73.2)	-27.211	17.554
Total	285			

* Numbers in parentheses are the 95-percent confidence limits.
** Standard deviation of the slope constant, b is ±2.946.

pressure in both the Series I and Series II experimental groups compared with controls. In terms of the magnitude of the overpressure of the reflected shock (psig), the Series I mice – those held for one hour at the pre-shot initial pressure before being returned to the Albuquerque ambient pressure – showed a fourfold increase in tolerance to be associated with a sixfold increase in the pre-shot ambient pressure. Series II animals – those returned to the Albuquerque ambient pressure immediately after exposure to blast – only exhibited a threefold increase in tolerance associated with the same sixfold increase in pre-shot ambient pressure.

The differences between the Series I and II data – shown clearly in Figure 5 – are of considerable interest and deserve several comments. First, in the experiments involving pre-shot pressures less than the Albuquerque ambient, blast tolerance was higher in the Series II than in the Series I mice. This means that mortality due to blast can be reduced by promptly pressurizing the animal after exposure to blast, as was the case for the Series II animals. This experience, consistent with the findings of Clemedson[10] and Benzinger[11] who demonstrated experimentally that early pressurization following a severe blast injury was beneficial and effective in reducing mortality, was not unanticipated since arterial air emboli, entering the circulation from the injured lung and known to be a prominent cause of early lethality in blasted animals, would be expected to decrease in size with pressurization and therefore become less hazardous to the animal.

Secondly and in contrast to the above results, the present study showed that blast tolerance of the Series I was greater than that of the Series II animals in all experiments involving pre-shot pressures above the Albuquerque ambient. These data mean that decompression carried out immediately after blast exposure, as was done in the Series II groups, increases lethality. One probable explanation is that blast-produced arterial emboli grow in size and therefore become a greater challenge to the animal. Another possibility is that more arterial emboli are produced by the decompression, a likely sequence of events should air trapping in the distal airways occur as a consequence of intra-bronchial hemorrhage, a not uncommon finding in blast-injured lungs.

Third, the Series I experiments in which the animals were held for one hour at the pre-shot ambient before being returned to laboratory pressures, no doubt are a more valid indication of the true variation in blast tolerance due to ambient pressure changes than are the Series II data. This seems so because (a) air emboli during the hold period have time to produce their biological effects, to decrease in size or to disappear from the circulation and (b) most individuals injured by blast are likely to be treated and held at the ambient pressure existing in the environment in which they were exposed. However, important exceptions to this statement are not improbable. For example, air evacuation of blast casualties could involve hazardous pressure changes. The post-exposure course of blast injuries occurring aloft in aircraft and during pressurized mining and tunneling operations could be worsened, improved or remain unchanged depending upon what pressure variations

occurred. Since control of the post-exposure pressure is not always impossible even in some emergencies, those who treat blast casualties should know two results of the present study; namely, (a) that the rate and range of decompression tolerated without demonstrable effect by control mice proved hazardous to blast-injured animals and (b) the rate and range of compression, producing no effect in controls when applied to experimental animals immediately after blast exposure, reduced mortality significantly.

Fourth, lethality-time data, limited in the Series I animals mostly to crude observation of body temperature and and the presence or absence of rigor, would aid further analysis of the differences noted between Series I and II mice. Such information will be forthcoming in future experiments since the end-plate of the shock tube has been fitted with an observation window.

Fifth, though the Series I data, as noted above, seem more applicable than the Series II results to most blast situations in "real life," it is well to consider the validity of the Series I findings further. In this regard, there are at least three matters of interest. The first is whether or not exposing an animal to a series of "sharp"-rising pressure pulses as exemplified in the lower left portion of Figure 1 bears any similarity to an exposure involving only a single "sharp"-rising pressure as might occur near a detonation in the open or in an open-ended or vented shock tube. Though these two situations, a single versus a repetitive pressure pulse, do seem different on the surface, the P_{50} figure of 31.0 psi (29.3 - 33.3), referable to an ambient pressure of 12 psi found in the present study, is not significantly different from those reported for mice in previous investigations with "long"-duration overpressures carried out at Albuquerque altitude; [2, 4, 12, 13] namely, 29.8 ± 0.8 psi for repetitive shock-tube pulses of 6 - 8 sec duration; 30.7 ± 0.6 psi for single shock-tube pulses of 400 msec duration; 29.0 ± 0.6 psi for single shock-tube pulses of 3 - 4 msec duration; 26.0 ± 0.4 psi for single high-explosive pulses of 2.1 msec duration; and 29.9 ± 1.1 psi for single high-explosive pulses of 1.3 msec duration. These data give power to the argument that it is the initial "sharp"-rising portion of a repetitive pulse of decreasing pressure that is definitive in producing lethality and not the second and subsequent oscillations, which indeed, seem to have little detectable effect.

Then, there is the question of possible differences in biological effect when using the shock-tube procedures described compared with actual free-field exposures to blast at various ambient pressures. For example, the technique developed represents an attempt to simulate a "real-life" blast situation in the laboratory, but the pressure-time variations in the shock tube — particularly over the immediate post-shot period — were hardly constant for the various experiments, represent departures from the ideal, and embody the potential for introducing variables into the experimental situation. Whether or not these may be significant, and if so, eliminated by improving the technique employed is not clear at the present time. However, appropriate work is under way in the laboratory and free-field experiments at different ambient pressures to check the shock tube data are being planned.

Also, since the P_{50} figures determined for the different ambient pressures represent an equal challenge to the animal – that is, the overpressures of 20.3, 31.0, 44.5, 55.3 and 91.8 psi above their respective ambients of 7, 12, 18, 24, and 42 psi are biologically equivalent – one searches for a constant parameter which, if approximately the same for each experimental group, might indicate consistency in the data, aid in their interpretation and increase confidence in the overall findings. That the pressure ratio, $\Delta P/P_i$, where P_i is the pre-shot ambient pressure and therefore the pressure inside the air-containing cavities of the body and ΔP is the blast overpressure and therefore the external pressure loading an animal, might be such a parameter is supported by the common-sense view that blast effects are sure to be importantly related to the magnitudes of the internal and external pressures, by the work of Haber and Clamann on the physics of rapid decompression,[14] by the findings of Luft and Bancroft[15] in biological studies of decompression and by White et al.[5] in blast studies wherein pulmonary lesions in dogs were correlated to the pressure ratio for nuclear blast waves inside shelters that rose in "steps" or in a "saw-toothed" manner. In this regard, the results of the present study are encouraging.

The pressure ratios shown in Table 2 are not constant, but ranged from 2.9 to 2.2 when the pre-exposure ambient pressure was varied from 7 to 42 psi. Blast tolerance, expressed this way, decreased by a factor of 1.3 or near 25 per cent when there was a sixfold increase in ambient pressure. If one uses the average pressure ratio of 2.5 shown in Table 2, it is possible to say that the P_{50} ratios only varied about 16 and 12 per cent above and below the average, respectively.

While experimental variations of these magnitudes are frequently noted in biological studies, the consistent and apparently not random decrease in the P_{50} pressure ratios with progressive elevation of the ambient pressures found in the Series I experiments not only stimulates one to search further for analytical understanding of the observed data to improve the grasp of etiologic mechanisms, but prompts a cautious approach to drawing general conclusions applicable to all mammalian species including man on the basis of the experiments reported here on mice.

Fortunately, it is now possible to say that similar studies have been completed using rats and guinea pigs. Preliminary analyses of the data show some randomness in the pressure-ratio figures and, in general, similar trends in blast tolerance with variations in the ambient pressure. Whether or not the pressure-ratio associated with such experiments is indeed a constant, with the differences noted indicating only "normal" experimental error and chance variations, cannot be stated now. But if results from future experiments with other species indicate that the LD_{50}'s can be expressed as multiples of the initial pressure, biological blast scaling as a function of ambient pressure will become a relatively simple matter. For example, man's tolerance (LD_{50}-24-hours) to "sharp"-rising overpressures of 400-msec duration has been calculated to be 50 psig from extrapolation of an interspecies correlation involving six different mammals.[2] Since the data

were compiled at an ambient pressure of 12.0 psia, the overpressure – normalized to the initial pressure – would be 4.2 atm. Consequently, to obtain the LD_{50} for "long"-duration air blasts for different ambient pressures, one may tentatively multiply the ambient pressure of interest by 4.2. Thus at sea level (14.7 psia), the calculated LD_{50} for man would be 62 psig; at 26,400 ft (5.2 psia) it would be 22 psig. It is well t emphasize the tentative and uncertain nature of these procedures, and it is no doubt premature to dwell on this topic further. Let it suffice to say that full understanding of biological blast scaling must await the results of future work.

Be this as it may, it is currently quite clear that the ambient pressure is indeed a physical parameter of major importance in specifying blast effects. Consequently, recording the local barometric pressure now needs to be considered a requirement in all quantative investigations of blast tolerance.

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